



DBS dysfunction mimicking transient ischemic attacks—a case report

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Received: 4 November 2019 / Accepted: 23 January 2020
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Abstract

We report on a patient with thalamic deep brain stimulation (DBS) for essential tremor who was admitted to a stroke unit with transient vertigo, dysarthria, and gait disturbance. Transient ischemic attacks were assumed but fluctuating neurological symptoms persisted until presentation to a DBS center. Here, unstable high monopolar impedances of the right-hemispheric electrode contacts were detected. Surgical revision revealed a fracture of the pocket adaptor connecting this electrode to the impulse generator. Replacement resulted in stable impedances and remitted the transient neurological symptoms. Emergency and stroke doctors should be aware of neurological symptoms induced by technical dysfunctions in DBS.

Keywords Deep brain stimulation · Impedance · Electrode dysfunction · Stroke · Transient ischemic attack

Introduction

Deep brain stimulation (DBS) is an established therapy for a variety of movement disorders, most notably Parkinson's disease, tremor, and dystonia. [6, 7, 9, 10] For this purpose, electrodes are implanted into the target structure and connected by extensions to an impulse generator (IPG). Electrodes provide 4 to 8 contacts which can be differentially activated to tailor the volume of tissue activated (VTA) at the electrode tip. During long-term DBS, stimulation parameters and consequently the VTA remain fairly stable. However, technical dysfunctions, most notably open and short circuits, can result in sudden changes of the VTA. [2, 3, 8, 11] In most of these cases, patients also experience loss of DBS efficacy and are consequently referred to DBS centers for troubleshooting. However, extension of the VTA to neighboring brain tissue as the result of short-circuited contacts has been reported to induce new neurological symptoms which were misdiagnosed

as stroke. [1] Here we report on a patient with DBS for essential tremor in whom the fracture of a pocket adaptor between extension and IPG induced transient neurological symptoms which were misdiagnosed as transient ischemic attacks.

Case report

The patient suffered from hereditary essential tremor of her hands since her early twenties. A significant worsening was observed in her fifties when the tremor also started to affect head and voice, later even the trunk. Medication was not efficacious. At the age of 72, implantation of a DBS system (electrode model 3389, Medtronic, extension model 7482, Medtronic, IPG Soletra, model 7426, Medtronic) for DBS of the left ventral intermediate nucleus (VIM) of the thalamus resulted in significant alleviation of the right-sided tremor. Six months later, another DBS system (electrode model 3389, Medtronic, extension model 7482, Medtronic, IPG Soletra model 7426, Medtronic) was implanted for additional right-hemispheric VIM DBS. Bilateral stimulation resulted in significant tremor reduction. Due to disease progression, the residual tremor worsened during the following years but stimulation continued to allow significant suppression. Voltage-controlled monopolar stimulation was used at 130 to 185 Hz and 60 μ s impulse width. On the left-hemispheric quadripolar electrode, the second contact was activated as anode for most of the time at 3.0 to 3.2 V. On the right-hemispheric electrode, the third contact was activated as anode for most of the time at

This article is part of the Topical Collection on *Functional Neurosurgery - Movement disorders*

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3.0 V. To reduce habituation, the patient was educated to stop stimulation during sleep. This prolonged the longevity of the IPGs. Hence, the first IPG replacement for low battery capacity was necessary after almost 9 years. The replacement was performed in a DBS center close to the patient's home address. Here, both single-channel IPGs were replaced by a dual-channel IPG (Activa PC, model 37601, Medtronic). The manufacturer (Medtronic) advises retunneling to allow both extensions to be located on the same side of the body. However, in the patient, the extensions were left in place. A dual-channel pocket adaptor (model 64002, Medtronic) was connected to the right extension and a single channel pocket adaptor (model 64001, Medtronic) was connected to the left extension. The reason for this specific surgical approach is unknown to the authors. Telemetric check of mono- and bipolar impedances 2 weeks after the replacement revealed normal mono- and bipolar impedances. Six months after the IPG replacement, the patient was admitted to a stroke unit for transient vertigo/dizziness, dysarthria, and deterioration of gait. Cranial computer tomography did not reveal ischemia or hemorrhage nor did extra- and transcranial ultrasound detect specific vascular pathology. Transient ischemic attacks were assumed due to paroxysmal atrial fibrillation and anticoagulation was switched from rivaroxaban 20 mg qd to apixaban 5 mg bid. Two weeks later, the patient presented to our DBS center because of continuing fluctuating symptoms. Telemetric control revealed variable mono- and bipolar impedances between and even within single measurements. Hence, monopolar impedances of contacts 8 and 9 in the first check were 1340 Ω and 1250 Ω and only seconds later at the next check 12,600 Ω and 12,800 Ω , respectively. Similarly, monopolar impedances of contacts 10 and 11 were 943 Ω and 1074 Ω , but bipolar

impedance of the same measurement 11,000 Ω . In the second measurement, only seconds later the bipolar impedance of these contacts 10 and 11 dropped again to 1722 Ω . The profound technical dysfunction necessitated surgical revision and revealed a fracture of the pocket adaptor (Fig. 1) to the right-hemispheric electrode. Replacement of the pocket adaptor resulted in stable tremor control and remitted transient neurological symptoms. Telemetric checks of the mono- and bipolar impedances immediately after surgical revision and at routine controls 3 and 6 months later revealed stable impedances within normal ranges.

Discussion

This case illustrates the importance to consider technical dysfunctions in DBS patients with new or fluctuating neurological symptoms. This is particularly relevant if sudden onset of new neurological symptoms is evaluated as vascular symptoms without immediate access to DBS experts. Misdiagnosis as stroke or transient ischemic attacks may result in harmful therapy decisions. [1] In our case, the cause of the dysfunction was a fracture of a pocket adaptor. Such fractures have been observed before and have led to additional recommendations by Medtronic concerning the implantation (U.S. Food & Drug Administration: class 2 device recall DBS pocket adaptor). Most importantly, the fracture did not result in complete disconnection of the active stimulation contact and, therefore, no stable loss of therapy. The fracture rather resulted in variable impedances and consequently variable VTAs at the activated electrode contact with fluctuating neurological symptoms. Technical dysfunctions including open

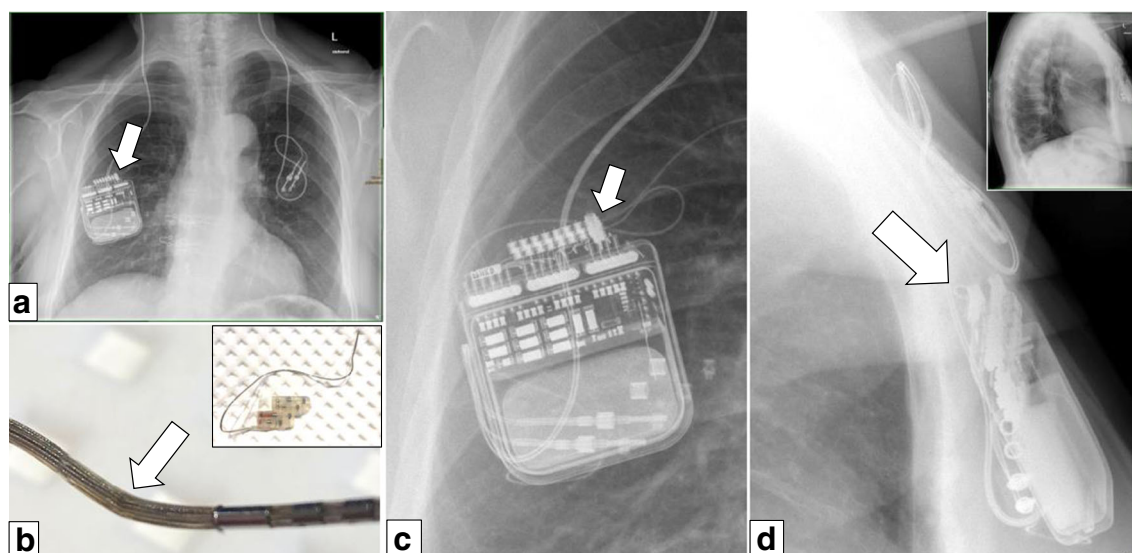


Fig. 1 a–d X-rays depicting the dual channel impulse generator (Activa PC, Medtronic, USA) and pocket adaptors (Medtronic, USA) to the right and left extensions. **b** The explanted right-hemispheric pocket adaptor. **c**

Magnification from **a**. **d** Lateral X-ray (inset → overview). The arrow individually points to the part in which a too sharp bend caused the fracture

and short circuits may also affect other hardware components and have been observed with increasing cumulative incidence during long-term DBS. [4, 5] As DBS is a life-long therapy applied in an increasing number of patients, the chance increases that DBS patients with new neurological symptoms are presented to emergency departments or stroke units prior to a DBS center. The most important issue is to consider technical dysfunctions in these patients particularly if symptoms can be related to neural structures in the vicinity of the DBS target. In our patient, the characteristic side effects in the vicinity of the VIM are dysesthesia, dysarthria, ataxia, and postural disturbance as described by the patient. “Cortical” neurological symptoms, in contrast, like aphasia, apraxia, or neglect, could not have been explained by DBS side effects and would point to another underlying pathology. If a technical dysfunction is considered, further evaluation with the specific physician’s programmer of the implanted IPG model is mandatory to determine mono- and bipolar impedances. The use of patient controllers can give a clue but may also be misleading without DBS experience. The patient controller cannot show impedance abnormalities but can be used to switch on and off stimulation. Lateralized stimulation-induced side effects will immediately disappear when stimulation is switched off. However, by switching off DBS, symptomatic relief of both electrodes is stopped and the reoccurrence or increase in motor symptoms may falsely suggest proper technical function. Hence, use of a patient controller is not sufficient to evaluate such technical dysfunctions and cannot substitute for a thorough evaluation in a DBS center.

Compliance with ethical standards

The patient has given written informed consent to publish her case.

Conflict of interest Niels Allert has received honoraria from Medtronic for lecturing and consulting and from Boston Scientific and Merz Pharmaceuticals for lecturing. Wei Jin and Xanthi Toulaki have nothing to disclose. Volker Arndt Coenen has received occasional honoraria and travel support from Medtronic (USA), Boston Scientific (USA), and St. Jude Medical (USA). He has ongoing investigator initiated trials with Medtronic and Boston Scientific with limited funding. He serves as medical advisor for CorTec (Freiburg, Germany).

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